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The Effects of Sleep Restriction on Executive Inhibitory Control and Affect in Young Adults

Kalina R. Rossa [a,b,*], Simon S. Smith, Ph.D. [a,b,c], Alicia C. Allan, M.Org.Psych. [b,c], and Karen A. Sullivan, Ph.D. [a,c]

[a] Clinical Neuropsychology Research Group, School of Psychology and Counselling, Queensland University of Technology, Brisbane, Australia
[b] Centre for Accident Research and Road Safety, School of Psychology and Counselling, Queensland University of Technology, Brisbane, Australia
[c] Institute of Health and Biomedical Innovation, School of Psychology and Counselling, Queensland University of Technology, Brisbane, Australia

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A B S T R A C T

Purpose: Young adults regularly experience restricted sleep due to a range of social, educational, and vocational commitments. Evidence suggests that extended periods of sleep deprivation negatively impact affective and inhibitory control mechanisms leading to behavioral consequences such as increased emotional reactivity and impulsive behavior. It is less clear whether acute periods of restricted sleep produce the same behavioral consequences.
Methods: Nineteen young adults (m = 8 and f = 12) with habitual late bedtime (after 22:30 h) and wake time (after 06:30 h) completed a range of objective and subjective measures assessing sleepiness (psychomotor vigilance task and Karolinska sleepiness scale), inhibitory control (emotional go/no-go task and a balloon analog risk task), and affect (positive and negative affective schedule). Testing was counterbalanced across participants and occurred on two occasions once following restricted sleep and once following habitual sleep 1 week apart.
Results: Compared with habitual sleep, sleep restriction produced significantly slower performance on the psychomotor vigilance task and higher subjective ratings of sleepiness on the Karolinska sleepiness scale. Sleep restriction also caused a significant decrease in positive affect but no change in negative affect on the affective schedule. Inhibitory control efficiency was significantly differentiated, with participants showing an increase in risk taking on the balloon analog risk task, but there was no evidence of increased reactivity to negative stimuli on the emotional go/no-go task.
Conclusions: Results suggest that even acute periods of sleep loss may cause deficits in affective experiences and increase impulsive and potentially high-risk behavior in young adults.

IMPLICATIONS AND CONTRIBUTION

Young adults regularly experience restricted periods of sleep, sufficient to provoke an increase in risk-taking behavior and a decrease in positive emotions. Further understanding of the role of sleep in young adults' emotional impulsivity may promote awareness of the protective value of sleep on "risky" behavior in this age group.

Sleep is a recuperative and restorative process that is imperative for the healthy development of cognitive, emotional, and social functioning. In adolescents and young adults, developmental changes such as the maturation of homeostatic and circadian sleep systems ensure that the timing of sleep is shifted toward a later bedtime, which, coupled with various psychosocial influences, may limit total sleep duration [1]. Crosscultural studies reveal that young adult university students sleep between 6.6 and 7.5 h on weeknights, with 70% sleeping less than 6 h at least one night a week [2–4]. Although "normal" sleep duration varies between individuals [5], sleep restricted to <6 h per night has been associated with measurable impairments in cognitive performance, vigilance, and affect [6,7]. In addition, a large population study found sleep quality to be rated as "poor" in 38% of young adults, with restricted total sleep time being a primary contributor to this rating [3]. The possibility that insufficient sleep contributes to behavioral, cognitive, and emotional

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* Address correspondence to: Kalina R. Rossa, Centre for Accident Research and Road Safety Queensland, QUT, Level 2, K Block, 130 Victoria Park Road, Kelvin Grove, Queensland 4059, Australia.
E-mail address: k.rossa@qut.edu.au (K.R. Rossa).

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disturbances in young people is supported by studies demonstrating that sleep restriction (SR) may lead to a range of cognitive [8], academic [2,9], behavioral [10,11], and emotional deficits (i.e., anxiety and depression [2,6]) in this group.

Despite the growing body of knowledge addressing the way in which SR may affect young adult cognition and affect, there have been few empirical studies assessing the effects of SR on higher-order inhibitory control and affective mechanisms in this age group. Inhibitory control mechanisms act to suppress immediate responses to incoming stimuli. The potential that in young adults restricted sleep may be adversely affecting these mechanisms is *indirectly* suggested by their behavior, which may include highly affective arousal, impulsivity, and risk taking [12,13]. In Australia, young adults are twice as likely as adults aged over 25 years to use illicit drugs, they have the highest rate of hospitalization for intoxication by alcohol, and they are more likely than other age groups to be charged with driving under the influence of alcohol and/or drugs [14]. It is likely that many factors contribute to such behaviors; however, the role of SR, through its potential effects on higher-order inhibitory control and affective mechanisms, is not well understood.

With a few exceptions [15,16], studies that have directly manipulated sleep duration to test effects on emotion regulation [6,17,18] or impulse control [19–21] support the notion that SR may be causing detriments to underlying inhibitory control and affective mechanisms. In one study, adolescents exhibited decreased positive affect and increased anxiety and catastrophizing following two nights of restricted sleep [6]. In another study, 55 h of continual wakefulness was associated with participants' inability to inhibit feelings of aggression toward others and an inability to behave in socially appropriate ways [18]. Furthermore, 36 h of continual wakefulness has been demonstrated to result in decreased affective inhibitory control efficiency in response to negatively valenced stimuli [19], and one night of total sleep deprivation [20] has led to an increased anticipatory pupillary response following presentation of negative picture stimuli, as opposed to positive or neutral stimuli. An implication of these studies is that sleep loss may decrease affective control and lead to heightened emotional impulsivity in certain social or environmental situations. While the link to risk-taking behavior is only demonstrated by one of these studies [21], these findings indicate that sleep loss has the potential to temporarily alter higher-order cognitive functions and emotion regulation in ways that may contribute to such behaviors. Understanding whether or not SR as is typically experienced by young adults leads to similar deficits is important, since sleep length is a modifiable factor, which could potentially contribute to a lowering of risk taking in young adults if increased.

The demonstrated links between sleep loss and temporary changes in cognition, emotion, and behavior are consistent with mild prefrontal lobe dysfunction [17]. The prefrontal cortex, which continues to mature up to the age of 25 years [22], is thought to be *most* vulnerable to the effects of sleep loss as it undergoes the greatest amount of metabolic change between sleep and wake states [23]. The prefrontal cortex is implicated in complex executive inhibitory and affective control via inhibitory subcortical connections with the amygdala, a limbic structure involved in the processing of affective goal-oriented actions [24]. It is thought that through these connections with prefrontal control centers, the amygdala acts to *modulate* approach or withdrawal actions towards affective stimuli in the environment [25]. Sleep may facilitate stronger prefrontal top-down control over the amygdala, allowing affective stimuli in the environment to be processed appropriately (i.e., with a level of control), whereas sleep loss leads to prefrontal cortical dysfunction, including reduced executive inhibitory control and limbic disinhibition (i.e., increased impulsivity to certain affective stimuli [25]). Thus the notion that SR could contribute to risky behavior in young adults is supported by this neurobehavioral model and remains to be tested objectively in this age group.

The present study investigated whether an acute *partial* SR paradigm would deregulate affective and inhibitory control mechanisms. This form of acute SR may typify the experience of young adults and provide results that are generalizable to their everyday experience. In order to maximize the effect of acute partial SR and circadian nadir on participant's performance, an early (05:00 h) testing time was chosen. We hypothesized that acute partial SR would (1) result in increased subjective and objective *sleepiness* (the desire for sleep) and that SR would; (2) deregulate affective and inhibitory mechanisms leading to a worsening of subjective affect; (3) produce behavioral consequences, specifically, increased reactivity toward negative stimuli; and (4) result in an increased propensity for impulsive, risk-taking behavior.

Methods

Subjects and conditions

Participants were recruited through advertisements in university environments. Following eligibility screening and study induction, 8 males and 12 females between the ages of 18 and 24 years (M age = 20.16 years, SD = 2.11) consented to participate. Participants were excluded if they were experiencing any of the following: self-reported sleep difficulties or disorders; significant health problems; taking illicit or prescription medications; drinking >3 caffeinated beverages a day; self-reported habitual bedtimes before 22:30 h and wake-up times before 06:30 h. Participants were either awarded credit toward their course of study or monetary compensation of AU$40.

Measures

Psychology Experiment Building Language [26]. Two tests from the Psychology Experiment Building Language (PEBL) test battery were used to objectively assess sleepiness and inhibitory control: the PEBL perceptual vigilance task (PPVT) and the PEBL balloon analog risk task (PBART). The tasks were presented in successive order on a Toshiba WT200 Windows Tablet 3G PDW03A-00G006 (Windows 7 OS). The computer tablet was attached to a viewing stand and connected to a standard keyboard (for registering responses). Basic noise reduction headphones were worn by each participant to reduce ambient noise interference.

The PPVT is a computerized version of the Wilkinson and Houghton [27] psychomotor vigilance task, which is a sustained attention, reaction time (RT) task sensitive to behavioral alertness [7]. Following sleep loss, psychomotor speed decreases due to reduced vigilance and arousal as evinced through slower RTs [7]. The PPVT required participants to press the "space bar" on the computer keyboard as quickly as possible in response to a colored stimulus (red circle) appearing on the tablet screen. RT in milliseconds was shown on the screen following each response. RTs greater than 500 ms are considered as "lapses" in behavioral alertness. The 10-minute test consisted of 120 trials of stimuli

presented at random interstimulus intervals ranging from 1 to 10 seconds.

The PBART is based on a validated, computerized test aimed to measure the construct of impulsivity through risk-taking propensity [28]. This 90-trial task involved accruing virtual money by inflating a virtual balloon. Five cents were accrued with each "click" of the balloon pump, and each "click" inflated the balloon by 1° on all sides. To retain money across trials, it needed to be banked before the balloon popped. Participants were not informed that there were three balloon types with different burst points, presented in randomized order. Participants could earn money by inflating the balloon, which they risked if the balloon popped, or bank money early, risking reduced earnings due to under inflation. Two risk indices were evaluated: (1) the total number of burst balloons per session (out of 90 presented) and (2) a cost–benefit ratio that represented the relative proportion of risk to the relative proportion of benefit obtained. A cost–benefit ratio >1 is indicative of greater cost incurred for less financial benefit (*see* [21] for details of cost–benefit ratio calculation).

The emotional go/no-go task [29]. This computerized task was administered using E-Prime, version 2.0., software. This task is an adaptation of the classic go/no-go task; however, it uses pictures of happy, neutral, and fearful faces as target ("go") and nontarget ("no-go") stimuli. The task consisted of six blocks of stimuli. Each block had 35 "go" and 13 "no-go" target facial expressions that were pseudorandomized across and between blocks to control for order effects. The go/no-go stimulus presentation ratio was 70:30. Instructions were displayed on-screen at the start of each block stating target and distractor expressions. Stimulus durations were 500 ms, with 2,000 ms interstimuli intervals. Participants were instructed to respond to targets as fast and accurately as possible (space bar press). To analyze the effect of SR on inhibition to negative stimuli, the mean number of false alarms (incorrect "no-go" trials) for fearful faces was used in statistical analyses.

The positive and negative affective schedule [30]. The positive and negative affective schedule (PANAS) was used as a subjective measure of positive and negative affects. These affective dimensions are distinct and independent of one another. Positive affect reflects the extent to which an individual feels enthusiastic, active, and alert, whereas negative affect reflects an individuals' level of subjective distress and unpleasurable engagement [30]. Therefore, a worsening in affect would be indicative of a decrease in positive affect and an increase in negative affect scores. The two PANAS subscales (one positive and one negative) each comprised 10 items. Participants indicated how much of a particular positive (e.g., "proud") or negative (e.g., "hostile") descriptor they had experienced that day using a five-point Likert scale ranging from 1 = very slightly to 5 = extremely. Composite scores for each scale range from 10 (minimum) to 50 (maximum).

Karolinska sleepiness scale [31]. This modified nine-item Karolinska sleepiness scale (KSS) was used to measure participant's subjective sleepiness at the end of each session. Participants rated their level of sleepiness by choosing the most appropriate statement that illustrated how "awake" they were feeling at that particular moment in time. Responses varied from 1 = extremely alert to 9 = very sleepy, great effort to keep awake, fighting sleep.

Actigraphy (Minimitter Respironics Actiwatch-II). Actigraphs (nondominant wrist-mounted accelerometers) were used to assess habitual sleep–wake behavior and sleep manipulation (partial SR) fidelity. Actigraphy is comparable with polysomnography as a valid and reliable measure of sleep–wake parameters, particularly sleep onset and wake times [32]. The actigraphs were set to record at medium wake sensitivity (40 activity counts per epoch).

Sleep timing questionnaire [33]. This retrospective questionnaire was used as an added control measure to assess participants' habitual bedtime and wake time on nights before work/study and the weekend. Convergent validity of the sleep timing questionnaire (STQ) in relation to actigraphy has previously been identified as .592 ($p < .005$) for bedtime and .769 ($p < .001$) for wake time [33].

Procedure

This study employed a within-subjects, repeated measures design with outcomes of the primary independent variable, SR being measured at two time points, once at 05:00 h (SR condition) and once at 09:00 h (non-SR condition), 1 week apart. The 05:00 h time of day was estimated to fall within the participant's habitual sleep period at a point close to their circadian temperature nadir (high predicted sleepiness following SR). The 09:00 h session was chosen to correspond with the rise in participant's circadian-mediated wakefulness. The order of testing conditions was randomized and counterbalanced across participants. Participants met with the experimenter 48 h prior to their first session and were fitted with actigraphs, which they wore until completion of the second session the following week. Participants were not allowed to drink caffeinated beverages for 6 h before testing.

For the SR condition, participants were asked to go to bed at their usual time and were woken up at 04:00 h by a phone call from the experimenter. Participants arrived at the laboratory in time to begin the experimental procedure at 05:00 h. Upon arrival, participants watched a movie for 20 minutes to counteract any residual physiological arousal effects from commuting. Once settled, participants completed the PPVT and PBART. Following a 5-minute break, participants completed the emotional go/no-go task, the PANAS, and KSS. For the non-SR condition, participants were asked to wake up naturally but not later than 08:00 h (at which time the experimenter called to confirm they had awoken). Participants arrived at the laboratory in time to begin the experimental procedure at 09:00 h. The same experimental procedure was used for the SR and non-SR conditions. The STQ [33] was administered at the end of the second session as an added control for the sleep manipulation. This study was approved by the Queensland University of Technology Human Research Ethics Committee; approval 1200000251.

Data analysis. To assess the fidelity of the acute partial SR manipulation, actigraphy recordings were reviewed for each participant and confirmed by sleep–wake information obtained from the STQ. Four participants' actigraphy recordings were unscorable due to technical failure in the recording device. STQ responses were evaluated for these participants. Actigraph data were recorded in 1-minute epochs, with rest intervals set using Actiware software (version 5.2), and sleep determined using the

"medium" sensitivity setting. Rest intervals were manually adjusted based on light data where discrepancies occurred.

Following actigraphy and STQ analysis, it was confirmed that one participant did not adhere to the required sleep–wake schedule (i.e., bed time after 22:30 h and wake time before 06:30 h). This participant was excluded from all further analyses, leaving a final sample size of 19. Table 1 lists actigraphy data. Following multivariate normality assessment, one outlier was located on the PBART, and these data were excluded from further PBART analyses.

A repeated measures multivariate analysis of variance (ANOVA) assessed the impact of SR (IV) on the combination of dependent variables.

Results

The multivariate ANOVA reflected a significant main effect of SR on the cognitive and affective dependent variables, Pillai's trace, $F(7, 11) = 4.330$, $p = .015$, partial $\eta^2 = .734$. Observed power to detect the effect was .871. To test for specific effects, follow-up repeated measures ANOVAs were conducted on each separate dependent variable. Results are listed in Table 2. Correlation coefficients between sleep and outcome variables are listed in Table 3.

Participants reported greater subjective (KSS, $p < .001$, $\eta^2 = .53$) and objective sleepiness (PPVT RT, $p < .05$, $\eta^2 = .24$) following the SR condition, relative to the non-SR condition. SR caused changes in positive (but not negative) affect. Mean positive affect ratings on the PANAS were significantly lower following SR, as opposed to non-SR ($p < .05$, $\eta^2 = .27$); however, mean negative affect ratings were not affected by SR and were consistently low on both occasions ($p > .05$).

SR increased participants' risk-taking propensity as evinced by a greater cost–benefit ratio ($p < .05$, $\eta^2 = .21$) and a greater number of burst balloons ($p < .05$, $\eta^2 = .46$). Participants' ability to inhibit responses to negative stimuli was not affected. The emotional no-go false-alarm rate for participants was similar following SR and non-SR ($p > .05$).

Discussion

This study aimed to establish whether acute partial SR would result in sleepiness and deregulated affective and inhibitory control mechanisms. This study sought to determine whether SR would result in a worsening of subjective affect and behavioral deficits, specifically increased reactivity to negative stimuli and increased propensity for impulsive, risk-taking behavior in young adults. First, an experimental manipulation check was performed to determine whether the partial SR paradigm was effective. This check showed that on average participants slept 3 h in the SR condition as opposed to their habitual 6.7 h (Table 1). Thus the manipulation was effective at producing a relatively mild but plausible manipulation of "one-time" sleep disruption (i.e., 70% of young adults sleep less than 6 h per night at least once a week [4]). As predicted, participants were sleepier at 05:00 h following acute partial SR, when compared with 09:00 h, and this effect was evident on both objective (PPVT) and subjective (KSS) measures. Following SR, subjective descriptors associated with mean sleepiness ratings on the KSS were "some signs of sleepiness" ($M = 6.74$, which previous research has found to be increased odds for sustaining injury in a car crash of approximately 4 [34]). Comparatively, following "normal" sleep, participants rated themselves as "rather alert" ($M = 4.89$), corresponding to an odds ratio of approximately 2 for sustaining injury [34]. These results are consistent with previous research documenting the negative effects of sleep deprivation on psychomotor speed and subjective assessment of sleepiness following sleep restricted to 6 h or less per night in adults [7].

Table 1
Actigraphic sleep characteristics

Sleep onset and wake times	$M \pm$ (SD)
Habitual sleep onset time	00:41 (00:30)
Habitual wake-up time	08:29 (00:48)
Sleep onset time prior to SR condition	00:42 (00:42)
Sleep onset time prior to non-SR condition	00:41 (01:03)
Wake-up time prior to SR condition	04:04 (00:17)
Wake-up time prior to non-SR condition	07:30 (00:34)
Sleep duration[+]	
Habitual sleep duration	405.26 (45.68)
Sleep duration prior to SR condition	181.93 (39.41)
Sleep duration prior to non-SR condition	344.63 (74.00)

N = 19; M = 24-h clock time; SD = "hours:minutes".
SD = standard deviation; SR = sleep restriction; [+] = sleep duration is in minutes.

The overall effect of SR on the combination of affective and neuropsychological variables was significant, explaining 73.4% of the variance in outcomes. The effect of SR on separate outcomes was, with two exceptions, consistent with the predication that sleepiness reduces function. Taking affect first, as predicted, experimentally restricting sleep produced changes in self-reported affect. While there was no change in response to ratings of negative affect between SR and non-SR conditions, there was a significant worsening of positive affect following SR. These results are consistent with previous findings [6], documenting a decrease in positive affect but no difference in negative affect following SR in adolescents and young adults. The large magnitude of effect for positive affect change in the present study ($\eta^2 = .26$) in spite of the relative brevity of sleep loss is important, given the association between sleep loss and increased anxiety in young people [6], and the links between impulsive, high-risk behavior, anxiety, and/or depressive symptoms [35]. The failure to find a worsening of negative affect following SR could be due to floor effects or due to differences in diurnal responsiveness of the PANAS positive and negative indices [30].

The hypothesis that SR would deregulate executive inhibitory control mechanisms was differentially supported. Previous literature has demonstrated that individuals are less able to inhibit responses to negative affective word stimuli following one night of sleep deprivation [19]. Our results contrast with these findings as there was no change in participants' ability to successfully inhibit their responses to negative stimuli on the emotional go/no-go task following SR (i.e., participants' number of false-positive responses did not vary between conditions). It is possible that these differences were a result of variability in processing affective word versus picture stimuli or that performance on this task may have been supported by other brain regions, blunting the effects of SR [36]. The paucity of studies utilizing this task in sleep research makes it difficult to compare results, particularly as the construct of emotion regulation encompasses a broad range of psychological dimensions [37].

The hypothesis that the propensity for impulsive risk-taking behavior would increase following SR was supported. On the PBART, participants were "popping" more balloons while sleepy and were also more willing to incur greater risk for less financial

Table 2
Means and SDs of outcome variables for SR and non-SR conditions and univariate repeated measures ANOVA results

Variable	Mean ± (SD)		*F*	*p*	Partial η^2
	SR	Non-SR			
KSS	6.79 (1.44)	4.89 (2.03)	20.54	.000**	.533
PPVT (RT)	411.85 (180.03)	374.01 (110.61)	5.71	.028*	.241
PANAS positive affect composite	21.00 (7.02)	26.42 (9.06)	6.473	.020*	.265
PANAS negative affect composite	15.26 (3.94)	14.89 (3.43)	.107	.748	.006
Emotional no-go (fearful faces FA)	8.42 (5.31)	7.0 (5.11)	1.027	.324	.054
PBART total burst balloons	50.11 (14.83)	41.79 (9.86)	14.268	.002*	.456
PBART cost–benefit ratio	1.84 (.71)	1.56 (.47)	4.63	.046*	.214

For PBART, N = 18; all other variables, N = 19; partial η^2 = effect size measure. ANOVA = analysis of variance; FA = false alarms; KSS = Karolinska sleepiness scale; PANAS = positive and negative affective schedule; PBART = PEBL balloon analog risk task; PPVT = perceptual vigilance task; RT = reaction time in milliseconds; SD = standard deviation.

* $p < .05$.

** $p < .001$.

Table 3
Correlation coefficients between sleep variables (PPVT and KSS) and outcome variables for SR and non-SR conditions

	SR condition		Non-SR condition	
	KSS	PPVT	KSS	PPVT
Outcome variables				
PANAS pos	–.727**	–.155	–.394	–.252
PANAS neg	.520*	.295	–.058	.083
Emotional no-go fearful faces FA	–.155	–.282	–.107	.451
PBART total burst balloons	.203	–.310	–.128	.237
PBART CBR	.476	–.132	.016	–.110

* = significant at the .01 level (two tailed); ** = significant at the .05 level (two tailed).

CBR = cost–benefit ratio; FA = false alarms; KSS = Karolinska sleepiness scale; PANAS neg = positive and negative affective schedule negative affect composite; PANAS pos = positive and negative affective schedule positive affect composite; PBART = PEBL balloon analog risk task; PPVT = perceptual vigilance task; SR = sleep restriction.

benefit as indicated by the increased cost–benefit ratio scores. The magnitude of this cost–benefit effect was "large" ($\eta^2 = .21$) and around half the size demonstrated previously following 75 h of total sleep deprivation ($\eta^2 = .39$ [21]). The number of trials on the PBART was 90, compared with 30 in a previous study [21]. A longer test has greater potential for time-on-task effects and is arguably more sensitive to sleep loss [36]. This may explain the large effect size relative to the amount of sleep loss incurred in the present study and emphasizes that a minimal degree of SR can result in large changes to the ways in which young adults evaluate risk-reward relationships. Whether these SR effects generalize to real-world situations is not demonstrated in this study; however, increased risk taking on the PBART has been associated with adolescent self-reported engagement in high-risk, impulsive behavior such as aggression, alcohol and drug use, and unprotected sexual intercourse [38].

Limitations

One limitation of this study is that actigraphy data were assessed for the week in between testing sessions; therefore, any uncharacteristic sleep variations occurring in the week before testing were not objectively assessed. Another limitation is that participants may have presented with a significant chronic sleep debt, given that habitual sleep duration was observed to be relatively low (6.7 h, see Table 1). This means that the results of this study may reflect the effects of *additional* acute sleep loss on a background of chronic sleep debt. While this caveat is not likely to be specific to this study, it should be borne in mind when interpreting results. Isolating the effect of acute sleep loss would require specific methodologies, such as extended and verified ad-lib sleep opportunity to ensure complete discharge of sleep debt [5]. Alternatively, a sleep extension condition may allow for better interpretation of findings in future studies.

The present study is the first of its kind to examine the effects of acute partial SR on the behavioral ramifications of deficient inhibitory control mechanisms in young adults. Although others have looked at similar outcomes [19–21], their methods used more extensive deprivation paradigms and, in some cases [20,21], mostly adult participants. This study demonstrated that an acute SR paradigm sufficient to produce sleepiness may significantly impact on affective experiences leading to a decrease in positive affect and cause sufficient deregulation of cognitive control that may manifest in increased impulsive or risky behavior. These findings are consistent with the broader epidemiology of risk-taking and impulsive behavior in this age group [12,39,40]. Further studies of the effects of restricted sleep on higher-order cognitive and affective functioning are needed to understand the neurobehavioral consequences of the relatively common occurrence of reduced sleep in the young adult population. A greater understanding may benefit clinicians, parents, and significant others in promoting awareness of the need for adequate sleep and for considering sleep as a primary factor in problematic behavioral profiles.

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